



Subarachnoid Hemorrhage

Perioperative Management

Patrick Neligan, W. Andrew Kofke
Department of Anesthesia, HUP

Learning Objectives

- To understand the pathophysiology behind subarachnoid hemorrhage
- To understand the steps taken to diagnose subarachnoid hemorrhage
- To understand the treatment options for SAH
- To understand the ICU management of SAH

Contents

- Pathophysiology of SAH
- Preoperative management
- Complications
- Anesthesia management
- Post-operative care

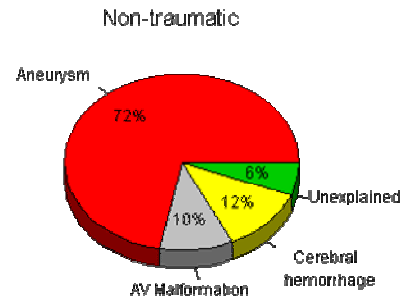
Pathophysiology

Subarachnoid Hemorrhage

Pathophysiology

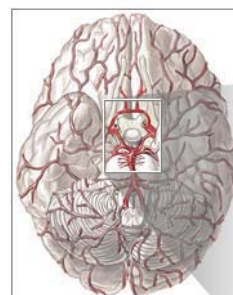
- SAH may be spontaneous or traumatic
- Spontaneous SAH are caused by
 - Cerebral aneurysms
 - AV malformations
- Uncommon causes – neoplasms, dural AVM, venous angiomas, infectious aneurysms

Subarachnoid Hemorrhage



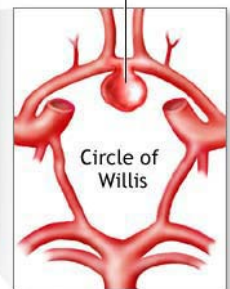
Aneurysms

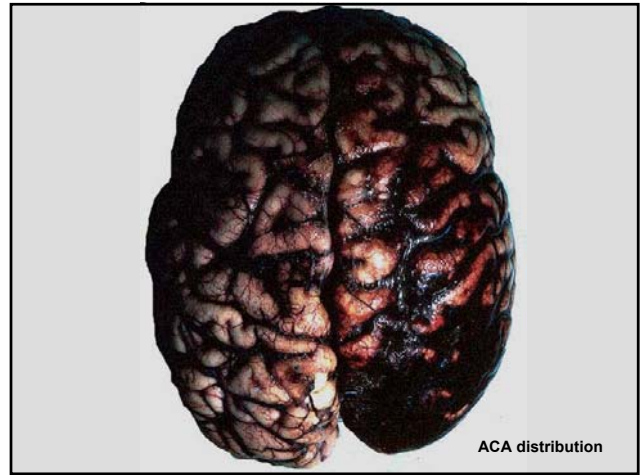
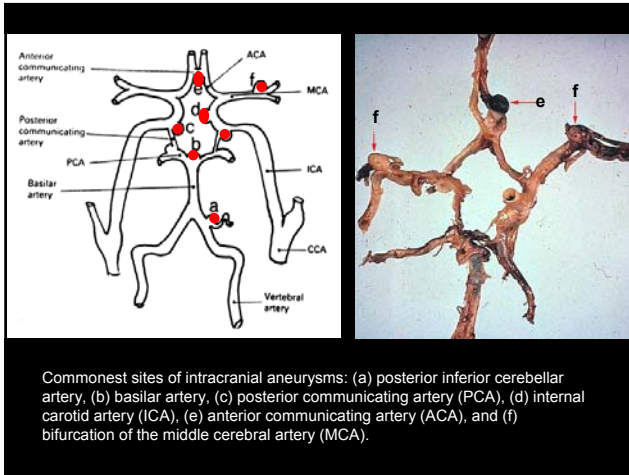
- 1-2% of the population have unruptured aneurysms
- Any aneurysm can rupture, although statistically larger (>1cm – 4%) aneurysms are more likely to do so.
- Women>Men, incidence increases linearly with age
- 10-15% of patients presenting with SAH have multiple aneurysms



Bottom view of brain and major arteries of the brain

Berry aneurysm on the anterior communicating artery of the brain



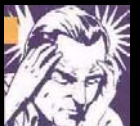


Diagnosis

Subarachnoid Hemorrhage

Premonitory Signs

- "Warning bleeds" are relatively common
- Sentinel headache 30-50%
- Early diagnosis prior to rupture will improve outcomes
- Unusual headache
- 50% of patients die within 48 hours irrespective of therapy



Clinical history
 Onset of headache: abrupt, maximal at onset, "thunderclap" headache
 Severity of headache: usually "worst of life" or very severe
 Qualitative characteristics: first headache ever of this intensity, unique or different in patients with prior headaches
Associated signs and symptoms
 Loss of consciousness*
 Diplopia*
 Seizure*
 Focal neurologic signs*
Epidemiologic factors
 Cigarette smoking
 Hypertension
 Alcohol consumption (especially after a recent binge)
 Personal or family history of subarachnoid hemorrhage*
 Polycystic kidney disease*
 Heritable connective-tissue diseases
 Ehlers-Danlos syndrome (type IV)
 Pseudoxanthoma elasticum
 Fibromuscular dysplasia*
Other
 Sickle cell anemia
 Alpha₁-antitrypsin deficiency
Physical findings
 Retinal or subhyaloid hemorrhage*
 Nuchal rigidity*
 Any unequivocal neurologic finding (focal or generalized)*

Risk Factors

Physical Findings

FINDING	LIKELY LOCATION OF ANEURYSM
Nuchal rigidity	Any
Diminished level of consciousness	Any (could result from possible complications of aneurysmal rupture: hydrocephalus, hematoma, or ischemia)
Papilledema	Any
Retinal and subhyaloid hemorrhage	Any
Third-nerve palsy	Posterior communicating artery
Sixth-nerve palsy	Posterior fossa*
Bilateral weakness in legs or abulia	Anterior communicating artery
Nystagmus or ataxia	Posterior fossa
Aphasia, hemiparesis, or left-sided visual neglect	Middle cerebral artery

Presentation

- "Worst headache in my life"
- Often accompanied by a period of unconsciousness – 50% do not awaken
- Neck stiffness, photophobia, headache
- Fundoscopy – subhyaloid hemorrhage

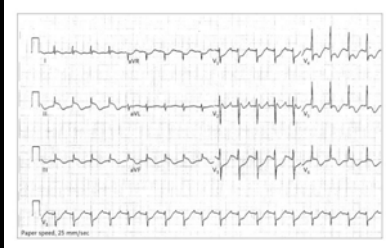


Diagnosis

- Painful 3rd nerve palsy
- Compression of the 3rd nerve by the PCA
- The pupil is dilated – different from diabetes which typically spares the pupil

ECG

- 20% have ECG evidence of myocardial ischemia
- ST segment elevation, T wave changes
- Due to high levels of circulating catecholamines



Hunt Hess Grade

Clinical Examination

GRADE	CRITERIA
0	Unruptured aneurysm
1	Asymptomatic or min headache & without neck rigidity
2	Mod-sev headache, neck rigidity, CN palsy
3	Drowsy, confused, mild focal deficit
4	Stupor, mild-sev deficit, decerebrate rigidity
5	Deep coma, decerebrate, moribund

CT Brain

- The initial study of choice is an urgent CT scan **without** contrast
- Sensitivity decreases with time from onset and with older resolution scanners.
- CT scan is 90% sensitive within the first 24 hours, 80% sensitive at 3 days, and 50% sensitive at 1 week.
- CT also can detect intracerebral hemorrhage, mass effect, and hydrocephalus.
- A falsely negative CT scan can result from severe anemia or small-volume SAH.

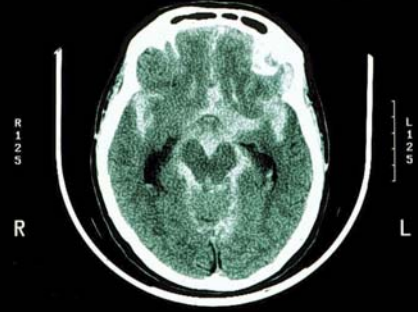
CT Brain

- Distribution of SAH can provide information about the location of an aneurysm and prognosis.
 - Intraparenchymal hemorrhage may occur with middle cerebral artery and posterior communicating artery aneurysms.
 - Interhemispheric and intraventricular hemorrhages may occur with anterior communicating artery aneurysms.
 - Outcome is worse for patients with extensive clots in basal cisterns than for those with a thin diffuse hemorrhage

Diagnosis

- If a “warning bleed” has taken place, the diagnostic sensitivity is 45%
- If the history is strongly suggestive, and the CT is negative, lumbar puncture is performed
- Xanthochromia is a classic sign, but not present early – look for equal or increasing blood in the sample tubes or D-dimers

Diffuse SAH



MCA aneurysm

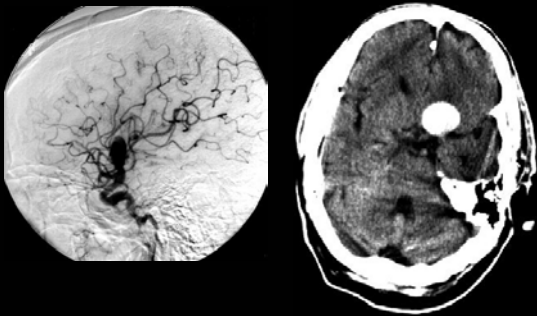


Blood in Sylvian fissure

Definitive Diagnosis

- Angiography (including MRA) – defines the source of the bleed.
- If multiple aneurysms are found – treatment targeted towards aneurysm adjacent to largest blood collection
- Sometimes there may be significant difficulty identifying the source

Giant Aneurysm



Complications

Early:
Rebleeding
Hydrocephalus

Complications

- **Hydrocephalus** may develop within the first 24 hours because of obstruction of CSF outflow in the ventricular system by clotted blood.
- **Rebleeding** of SAH occurs in 20% of patients in the first 2 weeks. Peak incidence of rebleeding occurs the day after SAH. This may be from lysis of the aneurysmal clot.
- **Vasospasm** from arterial smooth muscle contraction is symptomatic in 36% of patients.

Complications

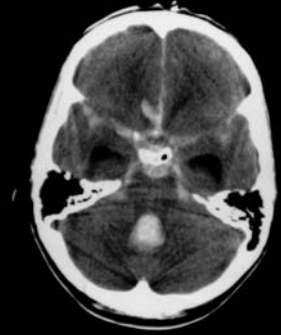
- **Neurologic deficits** from cerebral ischemia peak at days 4-12.
- **Hypothalamic dysfunction** causes excessive sympathetic stimulation, which may lead to myocardial ischemia or labile detrimental BP.
- **Hyponatremia** may result from cerebral salt wasting / SIADH
- **Nosocomial pneumonia** and other complications of critical care may occur.
- **Pulmonary edema** – neurogenic and non-neurogenic

Hydrocephalus

- Caused by obstruction of CSF flow by clotted blood
- Can occur early (EVD) or late (VP shunt)
- Careful with drainage – reduction in ICP can increase the risk of rebleeding

Hydrocephalus

- Temporal horns dilated
- Diffuse SAH
- Blood in the 4th ventricle
- Diffuse cerebral edema



Rebleeding

- Rebleeding occurs most frequently within the first 24 hours
- Up to 20% of patients rebleed within 14d
- Main preventative measure is control of blood pressure – beta blockers preferably
- Alternatively early clipping of the aneurysm allows hypertensive and hypervolemic therapy to prevent vasospasm

Hyponatremia

- SIADH
- Cerebral salt wasting

Treatment

Subarachnoid Hemorrhage

Treatment

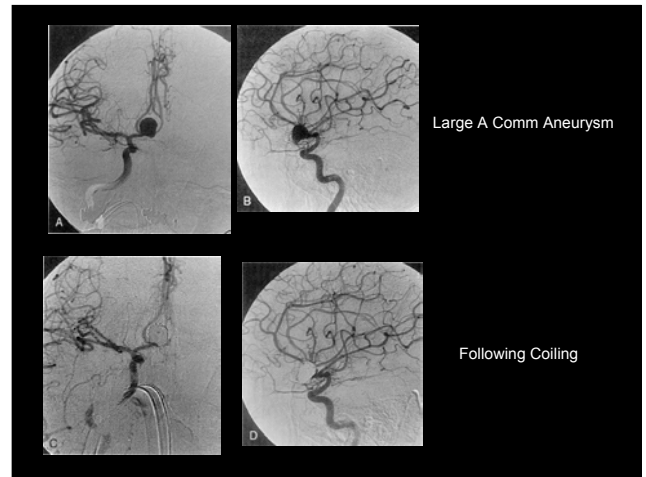
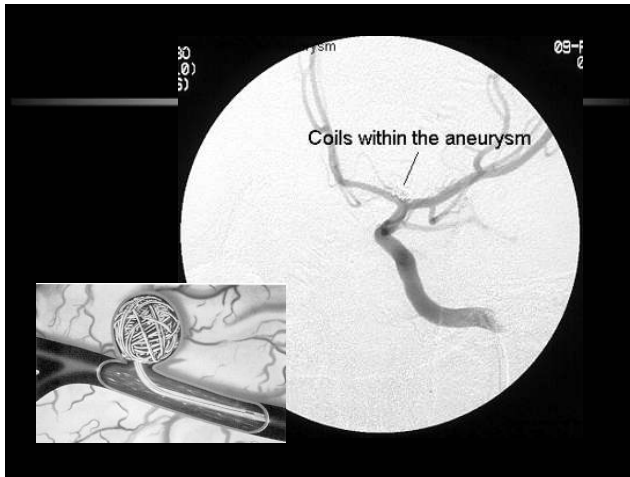
1. Identifying and treating the causative lesion, thus preventing re-bleeding
2. Treating hydrocephalus
3. Treating and preventing vasospasm

Early vs Delayed Surgery

- Early clipping – less rebleeding
- Higher incidence of vasospasm
- Worst time is day 7 to 10 (highest time for vasospasm)
- So – before 3 days, after 10 days

Surgery vs Coiling

- *International Subarachnoid Aneurysm Trial (ISAT) Lancet 2002*
- 2143 patients randomized to NS
- clipping (n=1070) or endovascular coiling (n=1073)
- Outcomes at 2 months and 1 year
- 23.7% coiling dependent or dead at 1 y
- 30.6% clipping at 1y (ARR 7% NNT 14)



Calcium Channel Blockers

- Nimodipine 60mg q6h x 24d
- Reduces:
 - Neurologic deficit
 - Cerebral infarction
 - Mortality

Anesthesia Management

Subarachnoid Hemorrhage

Anesthesia Management

Goals of anesthesia management:

- Prevention of rebleeding associated with acute hypertension (laryngoscopy, coughing etc.)
- Cerebral protection (cooling)
- Facilitation of surgery – “brain relaxation” (mannitol, propofol etc)
- Hypnosis, amnesia and analgesia

Preinduction Monitoring

- Routine anesthesia monitors
 - SpO₂
 - ECG
 - NiBP
- A Line
- 1 x large bore IV line (post induction)
- Temperature monitoring

Induction

- Objective
 - Hypnosis with tight control of blood pressure
- Accomplished with titrated:
 - Thiopental / Propofol
 - Fentanyl
 - LTA or IV Lidocaine
 - Vecuronium
 - Phenylephrine and antihypertensives available (Labetalol / Nicardipine / SNP)

Laryngoscopy and Intubation

- Slick and quick
- Give NMB 3+ minutes to work
- Ensure deep inhalational anesthesia
- Pre laryngoscopy bolus of propofol or lidocaine or esmolol

Postinduction monitoring

- Central line (+2 volume lines)
- Consider PA line if severe grade or preop ECG changes or HHH therapy being done
- Precordial Doppler
- SSEP
- EEG / BIS

Hypothermia

- Brain protection, prophylactic
- Numerous animal studies
- No randomized human studies in this setting
- Cooling blanket over-under
- Cool to 33 degrees until aneurysm clipped, then rewarm to 37 degrees

“Brain Relaxation”

- CSF drainage
 - EVD
 - Lumbar Drain – if EVD has not been placed
- Mannitol
- Furosemide
- Propofol
- Hyperventilation

Fluids & Electrolytes

- Electrolyte Problems are common:
 - Hyponatremia
 - SIADH
 - Cerebral salt wasting
 - Hypokalemia, hypomagnesemia and hypophosphatemia @ with diuretics

Fluids / Electrolytes

- Fluids – NaCl 0.9%, Normisol
 - Avoid all hypotonic fluids (including LR and especially glucose containing fluids) Be cautious of diastolic dysfunction (from myocardial ischemia)
- Replace mannitol urine loss
- Aim for normovolemia

Aneurysmal Hypotension

- Occasionally hypotension is required
- Proximal clip
 - Protection if long or EP's out
- Induced hypotension
 - Approach BP with isoflurane / propofol / labetalol or esmolol
 - Fine tune with SNP
 - Be Careful

Emergence and Recovery

- When clip on
 - Start rewarming
 - stop iv infusions of fentanyl and propofol
 - continue vecuronium
- When dura closed
 - Add N₂O if not already on
 - Gradually decrease isoflurane
 - Treat HTN with labetalol
- Extubate with first sign of wakefulness

Post-operative Care

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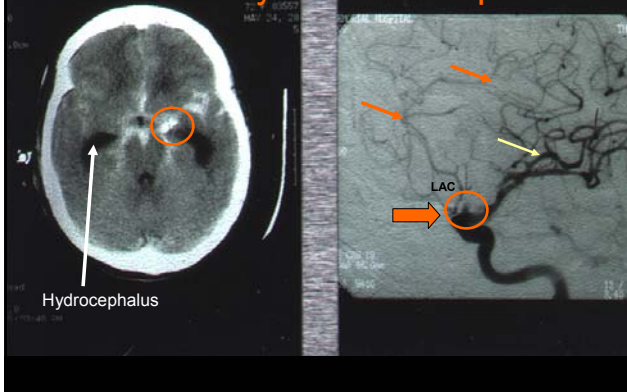
Blood Pressure Control

- Maintain systolic BP >130mmHg
- Use vasopressors if necessary – to maintain CPP, and reduce ischemic penumbra from vasospasm
- Generally avoid vasodilators (except calcium channel blockers)

Vasospasm

- Up to 33%
- Delayed until 48-72 h post SAH until 14d
- Associated with larger clots and increasing age
- Caused by local blood products
- Compensated for by increase in BP to maintain supply of nutrients
- Nimodipine / nicardipine

LAC Aneurysm + Vasospasm



Risk of Vasospasm

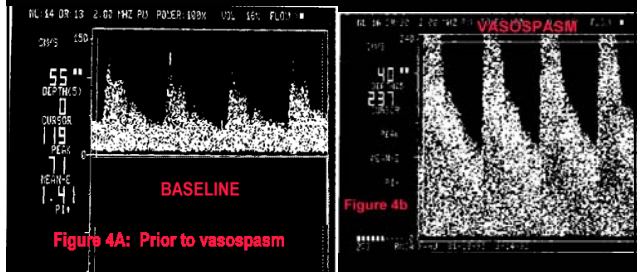
According to Clinical Grade

Hunt Hess Grade	%DIND
1	22%
2	33%
3	52%
4	53%
5	74%

Vasospasm Detection

- Neurologic exam
- Transcranial Doppler
- Angiography

Transcranial Doppler



Vasospasm

- Detection
- HHH therapy
 - Angioplasty
 - Papaverine
- Neuroradiology

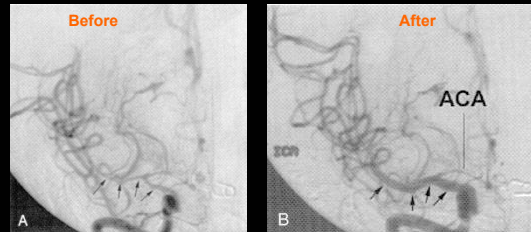
Vasospasm HHH Therapy

- Hemodilution
 - Hct 30-35%
- Hypertension
 - Phenylephrine / Norepinephrine
 - BP titration to CPP/exam
- Hypervolemia
 - Colloid/crystalloid
 - PCWP / CVO 12 or more

Vasospasm Neuroradiology

- Angioplasty
 - BP management during procedure
 - Reperfusion issues
 - Timing
- Papaverine infusion
 - Side effects
 - Repeated trips

Angioplasty for Vasospasm



Severe vasospasm of right internal carotid and middle cerebral artery (arrows) as well as spasm in anterior cerebral distribution in a patient after clipping of an anterior communicating artery aneurysm

Vasospasm and Catecholamines

- SAH: high catecholamine state
 - EKG changes
 - Myocardial ischemia
 - Neurogenic pulmonary edema
 - Spontaneous hypertension
 - Increased plasma catecholamines
- ? Role for Beta Blockers

